ance analysis before and after treatment with megestrol acetate.

Treatment with megestrol acetate leads to a better distribution of total body water with increased intracellular water component. Increased intracellular water is consistent with the increase observed in total cell mass. There was an increase in muscle mass and a small increase, not statistically significant, in fat mass. This increase in muscle mass would explain the increase in creatinine concentration observed in our patients.

Our results correspond to those of a study performed on a group of patients selected due to having experienced a significant weight increase after the administration of megestrol acetate. We can conclude that in this group of patients the increase in weight is not due to hydrosaline retention but rather, it is produced at the expense of an increase in lean mass, both of the cellular and muscular component. This finding is very significant if we take into account the association described between the increase in muscle mass and the better survival rate of the uraemic patient.[6]

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Table 1. Analysis of body composition by BIA before and after treatment with megestrol acetate.

	Baseline data	Post-treatment data	*P*
Dry weight (kg)	56.7±11.2	61.7±13.7	*P*=0.009
Total body water (l)	33.8±10.4	35.6±9.9	*P*=0.356
(% of body weight)	(57.9±8.8)	(57.7±5.9)	
Intracellular volume	49.9±5	56.5±5	*P*=0.01
(% total body water)			
Extracellular volume	50±5.6	44.9±4.8	*P*=0.01
(% total body water)			
Total cell mass (kg)	21.3±6.1	25±7.5	*P*=0.025
Fat mass (kg)	14.7±7.7	15.5±6.1	*P*=0.771
Muscle mass (kg)	26.7±7.6	30.8±8.9	*P*=0.033

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Effectiveness of haemodiafiltration with ultrafiltrate regeneration in renal failure due to multiple myeloma

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To the Editor:

Multiple myeloma (MM) is a clonal proliferation of plasma cells that leads to excessive production of a certain type of immunoglobulin or a fraction thereof. In 12-20% of patients, acute renal failure (ARF) occurs, mainly due to cast nephropathy (myeloma kidney) due to tubular damage. Survival depends on the recovery of renal function.

We present the case of a 63-year-old female, without relevant medical history, who was admitted with ARF secondary to MM (kappa light chain cast [LCC]) and remains dependent on renal replacement therapy (RRT) from diagnosis. Online haemodiafiltration was performed four times a week, in sessions lasting 240 minutes, maintaining residual diuresis (RD: 1000 cc/24 h) and serum creatinine levels around 5mg/dl.

The aim of MM treatment is to reduce the production of LCC with

chemotherapy and corticosteroids and/or autologous bone marrow transplantation. Coadjuvant treatment includes various techniques of extracorporeal clearance.

There are currently two types of RRT used as MM coadjuvant treatment: high cut-off haemodialysis (HCO) and haemodiafiltration with regeneration of ultrafiltrate (SUPRA-HFR). There are HCO studies in which these types of patients have a sustained rate of LCC reduction and recovery of renal function.[1] However, in another study in 6 patients, a higher rate of LCC reduction was not associated with the recovery of renal function.[2] SUPRA-HFR has recently been proposed for the removal of LCC in these types of patients.[3,4]

In our case, after obtaining LCC determinations and response to haematologic treatment, we decided to start SUPRA-HFR, three days a week, in 240 minute sessions.

We designed the following study to test the adsorption in resin of the LCC cartridge (kappa and lambda), of albumin and of beta-2-microglobulin with this haemodialysis technique, so that it may contribute to the removal of LCC, but without losing albumin. For this, we obtained blood samples every week pre-and post-haemodialysis and plasma water samples at minute 5 and 235 from the start of the technique, both pre- and post-cartridge.

The kappa LCC levels in serum were 105.19mg/l before the first session of SUPRA-HFR, and six weeks after the aforementioned treatment they were 61.18mg/l. The rates of reduction of each parameter both in blood and in plasma water are shown in the following two tables.

The results obtained show a mean blood reduction rate of 60% of kappa LCC, 32% of LCC lambda, 59% of beta-2-microglobulin and 3.5 % of albumin (Table 1). In plasma water, the rate of LCC reduction was much higher, both at the start and at the end of treatment: 98-99% of kappa LCC at the start and 80-94% at the end, in the first week of treatment (Table 2).

In spite of these results, the patient has not recovered renal function and she remains dependent on RRT, with blood creatinine of 7mg/dl and RD. She is awaiting an autologous bone marrow transplant.

With the results obtained, we can conclude that total saturation of resin does not occur, with a good rate of reduction of LCC being maintained (60% mean reduction of kappa LCC in serum), without the loss of albumin. Therefore, SUPRA-HFR is effective in the reduction of LCC as a coadjuvant treatment of MM.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Table 1. Reduction rates in blood.

Samples	1	2	3	4	5	6
Kappa	68%	53%	54%	64%	67%	55%
Lambda	35%	24%	32%	29%	46%	30%
Beta-2-microglobulin	64%	55%	47%	65%	66%	58%
Albumin	2.7%	2.7%	0%	3%	7%	6%

Table 2. Reduction rates in plasma water.

KAPPA	Sample 1	Sample 2
5'	99%	98%
235´	80%	94%

LAMBDA	Sample 1	Sample 2
5´	91%	92%
235´	72%	52%

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Hyporeninaemic hypoaldosteronism associated with membranous nephropathy: new hypotheses with regard to sodium retention in patients with proteinuria

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To the Editor:

Sodium retention is an expected situation in patients with proteinuria and hypoalbuminaemia. Though the conventionally proposed mechanism is based on the low plasma oncotic pressure produced by hypoalbuminaemia, a pathophysiological explanation that proposes a primary renal retention of sodium has recently been accepted.[1,2] This mechanism is independent of aldosterone. We present a case of membranous nephropathy with hyporeninaemic hypoaldosteronism.

A 43-year-old male was referred to our clinic with three grams of proteinuria a day. His history included common variable immunodeficiency that had required immunoglobulin infusion years before and idiopathic thrombocytopenic purpura untreated at present. The physical examination at the time of the consultation revealed blood pressure of 125/78mmHg, with a heart rate of 70 beats per minute and minimal oedema. The blood test showed the following parameters: haemoglobin 14.2g/dl, platelets 182,000 per μl, sodium 143mEq/l, potassium 4.2mEq/l, bicarbonate 25.2mEq/L, creatinine 0.73 mg/dl, urea 27mg/dl, albumin 3.4g/dl, total protein 6.0g/dl, cholesterol 247mg/dl, glycosylated haemoglobin 4.4%. The following values were found in urine: albuminuria 2868mg/day (albumin/creatinine 1017mg/g) sodium 228mmol/day and potassium 48 mmol/l. In the immunological study, anti-neutrophil cytoplasmic, anti-glomerular basement membrane, anti-double-stranded DNA and anti-nuclear antibodies were negative. Immunoglobulin and complement levels were also in the normal range. At that time it was decided to perform a renal biopsy, which revealed membranous nephropathy (Figure 1).

The patient was initially treated with renin-angiotensin-aldosterone system inhibitors, with proteinuria decreasing to 500mg per day. However, this treatment had to be discontinued due to hyperkalaemia. The transtubular potassium gradient was 4.71 and the suprarenal study confirmed hyporeninaemic hypoaldosteronism (aldosterone <1.6ng/l, plasma renin activity 0.1ng/ml/h), with a Synacthen test that stimulated cortisol and normal sex hormones.

We consider that this case offers new pathways for understanding the role of aldosterone in patients with proteinuria. There are currently two hypotheses that try to explain sodium and water retention in nephrotic syndrome: *underfill and overfill*. In the first of these, the mechanism is based on low oncotic pressure produced by hypoalbuminaemia accompanying the nephrotic syndrome.[1] As a result, there is renin and aldosterone stimulation, generating water and sodium retention. However, there is some controversy over this theory as the only cause of volume overload; as such, it has been proven that albumin administration in these patients does not produce an increase in natriuresis and that proteinuria *per se* does increase urine sodium excretion independently of plasma albumin.[3,4] Recently, a new hypothesis regarding primary sodium retention by the kidney has been developed and, as such, Svenningsen et al. have suggested that proteinuria includes the filtration of proteolytic enzymes capable of directly activating the collecting duct of the epithelial sodium channel, allowing sodium retention and thus inhibiting aldosterone.[2,5] Furthermore, in the nephrotic syndrome there is increased activity of phosphodiesterase in the collecting duct, allowing atrial natriuretic peptide and urodilatin degradation. The experimental administration of phosphodiesterase inhibitors reversed this positive sodium balance situation. This situation has also been demonstrated in patients with renal failure and cirrhosis.[6,7] Filtered proteolytic enzymes in patients with proteinuria include plasmin, which in normal conditions is not found in the urine. However, the conversion of plasminogen to plasmin by urokinase in these patients produces a direct action on the sodium channel gamma subunit in the collecting duct, whose mission is to inhibit said channel (and therefore mass sodium reabsorption). This causes primary sodium retention independent of aldosterone (which in these cases would be inhibited).[8]

Very few cases have been published on this situation.[9,10] The longest series includes 23 non-diabetic patients with nephrotic syndrome. Five of them had

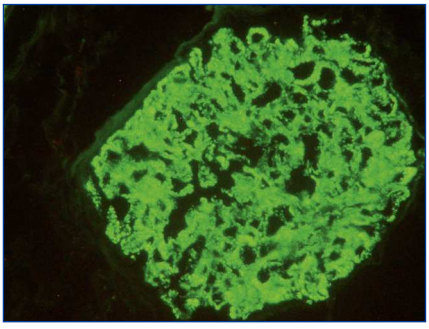

Figure 1. Renal biopsy.